THE

AMERICAN REVIEW OF TUBERCULOSIS
ABSTRACTS OF TUBERCULOSIS

VOLUME XXX DECEMBER, 1934 ABST. NO. 3

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Effects of Coal-Dust on Tuberculosis.—This experimental study covers several years' work with guinea pigs which are exposed to varying amounts of soft coal-dust before and after infection with tubercle bacilli of varying virulence. Due to circumstances beyond the control of the writers all the factors of the several groups are not exactly comparable but general summaries are presented before portions are repeated. In the first group 25 guinea pigs were divided into three sections which were exposed to coal-dust for 390, 150 and 630 days respectively. Some were killed immediately and others kept in normal atmospheres for varying periods, the longest of which was 988 days. Coal-dust was found to be distributed over the subpleural surfaces, on the ventral surfaces of the ventral lobes in thick lines, and along the paravertebral borders of both lobes. After long periods in normal atmospheres there was a marked tendency for dust to be found in geometric dots and lines in the above places. Dust filled phagocytes were in groups in the subpleural spaces. As time progresses after dust exposure, dust is found in increasing quantities in the connective tissue of the peripheral arterioles, bronchioles and veins. The lymph-vessels are normal. It is also found in the tracheobronchial lymph nodes outside the medullary channels and between the follicles. Dust is not eliminated by the air-passages. In the second group the animals were infected with R1 tubercle bacilli by inhalation infection and then exposed to coal-dust inhalation. The anthracosis was not appreciably modified by the coëxistent infection. This problem will be repeated. In the third group, R1 infection was superimposed on known anthracosis. Part of them were followed for 508 days. In neither the experimental animals nor the controls was there anything unusual about the tuberculous lesions. The fourth group consisted of 8 anthracotic guinea pigs and 8 normal controls infected with H60 tubercle bacilli. The controls died first. There was greater chronicity in the dusted animals and a greater tendency towards organization while cavity-formation and caseation were prominent in the controls. In the fifth group, 75 guinea pigs were infected with virulent tubercle bacilli, H37, and on the following day 50 were exposed to coal-dust-laden atmosphere. In the dusted animals death began to appear one week earlier than in the controls. Only 8 survived for 4 months and all died within 6 months. While only half the controls died in this period. Both groups showed generalized tuberculosis. The anthracotic group showed a slightly greater tendency to develop fibrosis. It can be stated that in anthracosis a nonvirulent tuberculous infection heals promptly but the evidence of protection against infection with more virulent tubercle bacilli is not so decisive, but is suggested by the character of the lesions rather than by their extent. In virulent infections there was little difference. There may be some protection by bituminous coal-dust against tuberculous infection.—*Experimental Inhalation of Bituminous Coal Dust and Its Effects upon Primary Tuberculous Infection in Guinea Pigs, L. U. Gardner, D. E. Cummings and G. R. Dowd, Journ. Indust. Hyg., November, 1933, xv, 456.*—(*J. F. B.*)

Pulmonary Sclerosis in Sulphur Miners.—Miners that work a long time in sulphur mines develop a form of pulmonary fibrosis which is not unlike silicosis. In

animal experiments it was ascertained that the disease can be produced by inhalation of sulphur without the addition of silicates. The clinical course and the roentgenological characteristics are presented in the form of 23 brief case-reports. It is claimed that this form of pneumonoconiosis, *theapneumonoconiosis*, according to Giordano, is quite frequent in miners exposed to sulphur dust.—*Weiterer klinischer und röntgenologischer Beitrag zur Untersuchung der Lungensklerose der Schwefelgrubenarbeiter (Theapneumoconiose nach A. Giordano), A. Ferrannini, Beitr. z. Klin. d. Tuberk., October, 1933, lxxxiii, 619.—(M. P.)*

Mineral Matter in Lungs.—Simplicity and completeness are the merits of this method of preparation of pulmonary tissue for the examination of mineral matter by transmitted light, dark-field illumination, and polarized light. About 30 or 40 gm. of lung are cut into small pieces and placed in a flask with 150 cc. of 20 per cent *Liquor trypsin* (Allen and Hanbury's) in a one per cent solution of potassium carbonate, and incubated at 37°C. In two or three days some of the turbid fluid is centrifugated, the deposit rendered slightly acid with sulphuric acid, washed in distilled water, and examined. This will contain some carbonaceous and other mineral particles, and in the various pneumonoconioses, particles associated with the respective diseases. The remaining fluid and lung are boiled for twenty minutes, cooled, 20 cc. of *Liquor trypsin* added, and reincubated for twenty-four hours. The fluid is accidified, centrifugated, the deposit washed, and examined. The deposit will contain the carbonaceous matter in addition to any other mineral present. Finally, to the remaining deposit 10 cc. of strong sulphuric acid and a crystal of potassium bichromate are added and boiled until clear. The tube is centrifugated, the deposit washed, and recentrifugated. This final deposit will contain the silica, insoluble silicates, and diatoms free from organic matter.—*Mineral Matter in Lungs, W. E. Cooke, Brit. M. J., September 9, 1933, no. 3792, 492.—(T. O. R.)*

Bilateral Spontaneous Pneumothorax in Pneumonoconiosis.—One patient is reported in whom bilateral spontaneous pneumothorax complicated pulmonary silicosis. At autopsy it was found that the silicosis was complicated by tuberculosis and that many subpleural emphysematous blebs were present. It is likely that the bilateral pneumothorax was caused by the rupture of some of these blebs. The originally partial pneumothorax on the right side was made complete by the perforation of a tuberculous cavity in the right lower lobe.—*Doppelseitiger Spontanpneumothorax bei Staublungenerkrankung, A. Abraham, Beitr. z. Klin. d. Tuberk., September, 1933, lxxxiii, 478.—(M. P.)*

Spontaneous Pneumothorax in Pneumonoconiosis.—This is a brief case report of three patients whose silicosis was complicated by spontaneous pneumothorax.—*Spontanpneumothorax bei Lungensilikose, Beitr. z. Klin. d. Tuberk., September, 1933, lxxxiii, 486.—(M. P.)*

Pulmonary Diseases and Noxious Gases.—One hundred and eighteen cases heard by the War Pensions Appeal Board in Auckland, New Zealand, were studied. The patients were divided into two groups, those exposed and those not exposed to war gases. A statistical analysis showed that asthma, clinical and roentgenographic evidence of pulmonary fibrosis, the "effort syndrome," and complaints referable to the chest occurred more frequently in the gas-exposed group. Pulmonary tuberculosis occurred approximately with equal frequency in the two groups. Pulmonary fibrosis is a late sequel and patients in whom it is present should avoid infection.—*The Incidence of Pulmonary Disease Following Exposure to Vesicant and Asphyxiating Gases, W. N. Abbott, Brit. M. J., November 11, 1933, no. 3801, 862.—(T. O. R.)*

Inhalation of Noxious Gases.—An adequate description is found in the literature of the symptoms and pathological changes resulting from the inhalation of noxious gases. However, the roentgenological findings are not so well defined. Films of the chest during the acute stage reveal a bilateral, evenly-distributed, parenchymatous involvement with scattered areas of consolidation. Some of the changes resemble those seen in the lungs secondary to cardiac decompensation. In favorable cases, all the changes show a tendency to clear rapidly. Three cases are reported. In a patient exposed to chlorine gas, the terminal finding was an acute ulcerative bronchitis, and, in 2 cases exposed to acid fumes, pneumonia was produced. One of the latter 2 patients recovered and the other developed an acute miliary tuberculosis. The relation of this complication to the irritant is unknown. These same 2 patients had pneumococci (type IV) in the sputum. It appears that the pulmonary structures are much less resistant to irritants of various kinds than are the nasopharyngeal structures. Experimental studies by Wood, Wollstein and

Meltzer, and Koontz and Allen indicate that irritative pneumonias differ from infectious pneumonias in that they are sterile and may manifest few symptoms.—*Pulmonary Changes from Inhalation of Noxious Gases, H. P. Doub, Radiology, August, 1933, xxi, 105.—(T. O. R.)*

Asthma and Tuberculosis.—The two diseases do not coëxist very frequently, according to the literature and to the author's own observation. Among 1,000 patients with tuberculosis, 14 had true bronchial asthma. In 13 of these 14 cases asthma preceded tuberculosis. Similar observations are reported by various authors. It is therefore unlikely that tuberculosis plays an aetiological rôle in the production of asthma. The clinical course of pulmonary tuberculosis is usually aggravated by asthma, while the course of asthma is not changed by pulmonary tuberculosis.—*Asthma bronchiale und Tuberkulose, A. Küpper, Beitr. z. Klin. d. Tuberk., February, 1934, lxxxiv, 255.—(M. P.)*

Asthma and Tuberculosis.—The association between tuberculosis and asthma may be summarized as follows: *1:* Only one asthmatic patient in a hundred gives any positive evidence of active tuberculosis. *2:* Only one patient with active tuberculosis in two hundred suffers from true asthma. *3:* When asthma does develop in a tuberculous subject the sensitizing agent is generally of an inhalant nature, and the asthma is relieved by its removal or by a course of specific desensitizing injections. In the rare cases in which the sensitizing agent is bacterial in nature it is more often a secondary invader than the tubercle bacillus.—*Tuberculosis and Allergic Diseases, G. W. Bray, Brit. J. Tuberc., January, 1934, xxviii, 7.—(E H. K.)*

Effects of Inhaled Adrenalin.—The effects of inhaled adrenalin were studied by spirographic methods in 13 patients suffering from bronchial asthma. It was found that the pulmonary ventilation was improved after the medication. In some of the cases this was achieved by a decrease of the pathologically increased residual air. In another group of patients, the residual air was not changed but the total pulmonary capacity increased. In all cases the respiratory rate and the minute volume of respiration decreased. The systemic effects that adrenalin causes on parenteral administration were absent after administration by inhalation.—*Untersuchungen über den Einfluss inhalierten Adrenalins auf die Lungenventilation beim Asthma bronchiale und über dessen Allgemeinwirkung, K. Lageder, Beitr. z. Klin. d. Tuberk., October, 1933, lxxxiii, 605.—(M. P.)*

Massive Atelectasis.—By massive atelectasis is meant the collapse of one or more lobes of the lung. The cause of this collapse is generally accepted to be the complete occlusion of a bronchus. Roentgenological examination provides the most reliable procedure by which a correct diagnosis may be established. Atelectasis must be differentiated from infarct, pneumothorax, subdiaphragmatic abscess, embolism, pleural effusion, fibroid tuberculosis, and pneumonia. The last-named condition offers, by far, the greatest difficulty. In atelectasis, the diaphrgam is elevated on the affected side and the heart, trachea, or mediastinal structures are displaced to a greater extent than is found in pneumonia.—*The Roentgen Diagnosis of Massive Atelectasis of the Lung, J. B. Johnson & C. F. Crain, Radiology, 1933, xxi, 388.—(T. O. R.)*

Atelectasis.—Atelectasis as a complication of pulmonary tuberculosis has been emphasized only during recent years. It is due to bronchial obstruction, which results from aspiration of a caseous plug, haemorrhage with clot-formation, plugging of a bronchus with thick tenacious sputum, pressure from an enlarged lymph node, or possibly contraction of a cicatrix. Its onset is usually sudden and with few symptoms. It may, however, occur successively in smaller or larger areas and thus give the impression of a gradual development. Roentgenological examination shows a marked opacity over the involved lobe or lobes with a displacement of the heart, trachea, mediastinum and diaphragm toward the affected lung. The interspaces may be narrowed and the spine may be curved in the opposite direction. The treatment of atelectasis which complicates tuberculosis depends upon the presence of cavitation in the involved lung. Collapse therapy is indicated in cases in which the closure of cavities is not obtained by this complication alone. In tuberculosis, atelectasis tends to remain permanent. The lung becomes fibrotic.—*Atelectasis as a Complication of Pulmonary Tuberculosis, E. A. Gatterdam, Radiology, September, 1933, xxi, 251.—(T. O. R.)*

X-ray Changes in Atelectasis.—Atelectasis has certain roentgenological characteristics which differentiate it from other conditions. Roentgen phenomena of atelectasis fall into two classes: (1) increase in the density of the shadows of the involved portion of the lung, (2) displacement of the heart and other mediastinal structures to-

ward the side of involvement, with elevation of the diaphragm on the affected side. Acquired atelectasis in bronchial foreign-body, bronchial neoplasm, postoperative massive collapse, pneumonia, tuberculosis and asthma, is due to intrabronchial plugging; and in enlarged tracheobronchial lymph nodes, aneurysm, mediastinal new growth and extrabronchial pulmonary neoplasm it follows occlusion of the bronchus from extrabronchial pressure. The close association of atelectasis and bronchiectasis is not generally recognized. In tuberculosis, atelectasis may disappear spontaneously leaving no sign, or it may become chronic and end in bronchiectasis.—*Significance of Roentgenologic Changes in Differential Diagnosis of Atelectasis, Willis F. Manges and John T. Farrell, Jr., Am. J. Roentgenol. & Rad. Ther., October, 1933, xxx, 429.*—(*E. R. L.*)

Acute Yellow Atrophy in Tuberculosis.—This is a case report of the occurrence of acute yellow atrophy in a woman, 27, while undergoing, with satisfactory response, sanatorium treatment for a moderately advanced pulmonary tuberculosis. In onset, clinical course and autopsy findings there was a striking similarity to those cases reported in the literature in which death followed the administration of sanocrysin. Sanocrysin had not been given in this case. The cause of the acute yellow atrophy was not determined.—*"Acute Yellow Atrophy" in Pulmonary Tuberculosis, A. Lynn & J. S. B. Mackay, Lancet, January 20, 1934, ccxxvi, 130.*—(*B. T. McM.*)

Bronchiectasis.—One hundred and six confirmed cases of bronchiectasis were studied for a period of three to six years in regard to their general and local condition and the character of their sputum in relation to various types of treatment. The patients were divided into "dry" cases in which there was no expectoration, "septic" cases in which the sputum was purulent, and "foetid" cases in which the sputum was copious and foul. Cases with occasional sputum, classed "simple," ran the same course as the persistently septic and were, therefore, not separated from them. This classification, although not exactly coinciding with the anatomical varieties, is more directly valuable for a prognostic and therapeutic approach. The total mortality of 38 per cent emphasizes the severity of the condition. Bronchiectasis with only medical treatment is an extremely fatal disease. Of 49 patients, 23 are dead, 9 totally incapacitated, and only 4 of the remainder are "dry" five years after the diagnosis. Of 14 foetid cases, only 2 patients survived, and of these one is totally incapacitated. In contrast to this group, the 57 patients who had some form of surgical intervention fared better. Of these, 18 are dead, 18 incapacitated, and 20 are well. The surgical treatment does not appear to be as successful in the cylindrical as it is in the saccular types, which is the reverse observation of that with medical treatment. Thirty-six cases of the total 106 were reëxamined with lipiodol in order to determine the liability and direction of extension of the pulmonary involvement. It appears that with medical treatment spread is almost certain in a very few years, and the involvement tends to become bilateral; whereas with surgical intervention, apart from lobectomy, a gradual extension of sacculation to other zones on the treated side occurs but contralateral spread is unusual. The results of the operative procedures were analyzed. Artificial pneumothorax has proved to be ineffectual and is now used only as a preliminary to lobectomy. Pneumothorax combined with phrenic evulsion appears to give slightly better results than either alone. Phrenic evulsion alone is of doubtful value and is contraindicated if lobectomy is feasible. Thoracoplasty should never be performed for unilobar disease but may result in marked symptomatic improvement in patients with advanced unilateral involvement. Lobectomy is the only procedure which produces a radical cure. It depends for its success upon the early diagnosis of the condition and expert supervision of the pre- and post-operative care. In order to secure early diagnosis, all doubtful cases of haemoptysis and cough, in which tuberculosis has been excluded, should be X-rayed after the instillation of lipiodol. Lateral roentgenograms are indicated in those cases in which anteroposterior screening shows an apparent unilobar distribution of the opaque medium.—*Bronchiectasis: Diagnosis and Prognosis in Relation to Treatment, F. C. Roles & G. S. Todd, Brit. M. J., October 7, 1933, no. 3796, 639.*—(*T. O. R.*)

Congenital Bronchiectasis.—A case of congenital bronchiectasis in a 11 year-old boy is reported. That the condition was congenital in origin was substantiated by the clean-cut circular type of bronchiectatic dilatations which are not found when inflammatory lesions are the cause, by the ability to produce a complete artificial pneumothorax which is good evidence that past inflammatory and fibrotic lesions were not the causative factor, and by the absence of pigment on the pulmonary surface as seen with the thoracoscope which is evidence in a

town-dweller that the alveoli in that side are absent.—*Congenital Bronchiectasis (With Special Plate), H. V. Morlock and A. J. S. Pinchin, Brit. M. J., October 28, 1933, no. 3799, 780.*—(*T. O. R.*)

Bronchiectasis and Situs Inversus.—Four cases are described in some detail in which bronchiectasis, situs inversus and chronic inflammatory disease of the nose and the sinuses coëxisted. It is likely that the bronchiectases in these patients were congenital. The frequency of situs inversus is, according to the Medical Clinic of Leipzig, 5 in 63,377 cases; according to the Pathological Institute of Leipzig, 3 cases in 22,402 autopsies; according to the Mayo Clinic, 1 in 34,000. It is much more difficult to establish the actual frequency of bronchiectasis. According to the material of the Eppendorfer Hospital in Hamburg 501 cases of bronchiectasis were found among 34,000 autopsies. The relative frequency of bronchiectasis and situs inversus is such that the coëxistence of the two conditions is probably not fortuitous, but that it represents a characteristic syndrome.—*Zur Pathogenese der Bronchiektasien, M. Kartagener, Beitr. z. Klin. d. Tuberk., September, 1933, lxxxiii, 489.*—(*M. P.*)

Pathogenesis of Bronchiectasis.—The literature contains many statements about congenital bronchiectasis and about the frequency with which bronchiectasis occurs repeatedly in the same family. A critical analysis of the literature does not reveal much evidence for a definite hereditary factor. The clinical histories and the roentgenological findings of two pairs of siblings of bronchiectasis are reported in detail. The conclusion is reached that even when the hereditary or congenital nature of the disease cannot be proved a certain hereditary weakness of the bronchial structures may be assumed. Such a congenital debility of the bronchial walls may be present in the following three groups of bronchiectasis: (1) in strictly congenital bronchiectasis; (2) in bronchiectases which develop slowly and insidiously without inflammatory or mechanical irritation,—the so called idiopathic type of Bard; and (3) in bronchiectases of an inflammatory infectious nature in which the congenital weakness of the bronchial wall accounts for the progressive affection of the bronchi.—*Zur Pathogenese der Bronchiektasien, M. Kartagener, Beitr. z. Klin. d. Tuberk., December, 1933, lxxxiv, 73*—(*M. P.*)

Iodized Oil in Bronchiectasis.—Studies were made to determine the disposition of iodized poppy-seed-oil after pertracheal instillation into the bronchiectatic lung. In two cases of far-advanced and long-standing but localized bronchiectasis, expectoration decreased following pertracheal injections of iodized oil, but the foul odor and signs of sepsis were relieved only after lobectomies, which were done by ligation of the pedicle and cauterization. Sections of the diseased lobes were removed just before ligation of their pedicles. In one case twenty-two days, and in the other case eighty-two days had elapsed since iodized oil was injected. Microscopic study of the sections revealed diffuse overgrowth of connective tissue, chiefly in the fibroblastic stage, and a marked inflammatory reaction throughout. The bronchial lumina were irregular, with hyperplasia here and loss of mucosa there. Bronchioles were uniformly collapsed. In some areas there were large collections of oil in the bronchi. Oil droplets were found in all types of cells of the parenchyma and the bronchi, particularly in the large mononuclear cells of the alveolar walls, and also outside of cells in the dense fibrous tissue. Apparently the ingestion of the fat particles is not essentially phagocytosis, but is rather due to the natural affinity of cells for fat. Retention of oil in the tissues might be of therapeutic value through the slow liberation of iodine. Failure of oil to enter some of the bronchiectatic sacs explains its incomplete therapeutic effect in the saccular type and its greater benefit in the mild cylindrical type of bronchiectasis. Each of 11 guinea pigs, after ether anaesthesia, received 1 cc. of iodized oil by intratracheal injection through a neck incision. Animals were killed on the third, fourth, seventh, eleventh, fourteenth, and twenty-eighth days following. In four, death occurred on the thirty-ninth, forty-first, forty-fourth, and fifty-fifth days. The lungs of these guinea pigs were sectioned and the lung of one which had not received oil was sectioned for comparison. From these experiments it was concluded that (1) the injection of iodized oil causes multiple areas of atelectasis, apparently due to the plugging of bronchi and bronchioles; (2) the areas of atelectasis are always accompanied by inflammation; (3) oil droplets remain for a longer time in areas of atelectasis than they do in well-aërated lung; (4) oil droplets are quickly taken from the spaces by the tissues and may remain in the alveolar walls or may be carried for some distance, as shown by the finding of oil droplets in the pleura. The main bronchi to the right lower lobes of 8 rabbits were ligated with absorbable sutures under ether anaesthesia, in order to produce an infection in the lung simulating bronchiectasis. Those

rabbits surviving beyond the sixth day received intratracheal injections of iodized oil. The ligated lobes became necrotic to a greater extent than occurs in typical bronchiectasis. Conditions simulating bronchiectasis were also seen in the upper and middle lobes, which were collapsed temporarily during the ligation operation. Abscess-formation without bronchiectasis occurred in the contralateral lung, and from this it is inferred that atelectasis is necessary to the production of bronchiectasis. It appeared that the oil remains for a longer time in an abscessed adherent lobe than it does in a more normal lobe. It is assumed that expulsion of oil through the trachea is aided by free mobility of the lung. Some of the oil takes the path of the lymphatics.—*Iodized Oil in Bronchiectasis, Including a Study of Two Cases Following Lobectomy, J. A. Weinberg, Arch. Surg., September, 1933, xxvii, 545.*—(*R. M. F.*)

Bronchoscopy in Bronchiectasis.—Bronchoscopy is considered an important aid in the diagnosis of bronchiectasis. It is much more important and necessary from a therapeutic standpoint, being the means by which haemorrhage may be controlled or purulent secretion aspirated in patients who are unable to drain sufficiently by postural methods. Bronchoscopy's greatest efficacy in this disease is found in the treatment of those cases with a secondary pneumonitis about the areas of bronchiectasis. Here bronchoscopic aspiration may be the only effective method to prevent the spread of the inflammatory reaction with possible abscess-formation and death.—*The Importance of Bronchoscopy in Bronchiectasis, H. P. Marvin, Ann. Int. Med., January, 1934, vii, 903.*—(*B. T. McM.*)

Collapse Treatment of Bronchiectasis.—The efficacy of artificial pneumothorax as a means of healing ulcerative tuberculosis of the lungs has been demonstrated. The procedure has, however, been given a very limited trial in bronchiectasis despite the fact that this is a disease which does not recover spontaneously, but causes serious disability and terminates fatally. In 1928 Whittemore and Balboni found no more than 93 cases treated by pneumothorax in the world literature. Ninety cases of bronchiectasis seen between 1920 and 1931 are reported; 31 were not treated by pneumothorax, either because of their serious condition or because they refused treatment. In 59 cases pneumothorax was attempted; in 22, or 37 per cent, it was unsuccessful because of pleural adhesions. Of the 37 cases whose lungs were collapsed, 17 were not benefited. In 6 of these collapse was ineffective because of diaphragmatic adhesions. In one case drainage from a cavity was interfered with, while 10 cases either had bilateral disease or gangrene of the lung and died. The remaining 20 cases, one third of the total treated, were rapidly freed from symptoms, although 7 of these could be kept so only by the maintenance of pneumothorax. In 13 cases the cure was complete and has lasted for periods of 3 to 8 years. In 11 of the 22 cases with adherent pleurae, phrenicectomy was performed: three cases recovered completely while four others were much improved. The success of collapse therapy is attributed to the provision of efficient and thorough drainage of the bronchial tree which paves the way for the healing of the bronchial, or more precisely, the bronchopneumonic infection. The symptoms of bronchiectasis are due to infection and not to dilatation. A noninfected bronchiectasis is an anatomical abnormality, not a disease.—*Collapse Therapy of Bronchiectasis, E. Rist, Ann. Int. Med., October, 1933, vii, 417.*—(*R. D.*)

Collapse Treatment of Bronchiectasis.—Two case histories of bronchiectasis are reviewed, to illustrate the unfavorable influence collapse procedures may exert against the successful outcome of a subsequent lobectomy. A paralyzed diaphragm removes the principal factor in expanding the lung and prolonged artificial pneumothorax so anchors the remaining lobe that it will not expand quickly. Early expansion of the remaining lobe is essential, however, so that the pleural space is obliterated before infection sets in.—*Collapse Therapy in Bronchiectasis, H. P. Nelson, Brit. M. J., January 13, 1934, no. 3810, 58.*—(*T. O. R.*)

Cystic Disease of Lung.—For two and a half years the authors have observed a 3-year-old boy, whose entire left thoracic cavity was filled with a balloon-like cyst. His condition varied from that of threatened asphyxia to that of restful normal breathing, relief being effected only by aspirating from 8,000 to 10,000 cc. of air from this sac every four to six hours. The case is of interest because the patient has made an apparent recovery from a condition that usually terminates fatally when it occurs at this early stage of life, and the results obtained seem to suggest some worth-while therapeutic measures. Congenital cystic disease of the lung occurs in two forms: (1) the solitary cyst, which is usually large, and (2) multiple small cysts, which involve a part or all of a lung. Unexplained attacks of dyspnoea and cyanosis with the signs of pneumothorax

should lead one to suspect such a condition. The roentgenograms will in most cases show a large air-sac that often replaces the normal lung tissue of an entire lobe or lung, suggesting at first glance partial or complete pneumothorax on the affected side. The differential diagnosis includes pneumothorax, diaphragmatic hernia, and foreign body in one of the larger bronchi with a resulting atelectasis. Roentgen studies after injections of lipiodol will often be of aid in making a correct diagnosis, and it is possible that it has some therapeutic value.—*Congenital Air Cyst of the Lung: Report of a Case, C. V. Croswell & J. C. King, J. Am. M. Ass., September 9, 1933, ci, 832.*—(*G. L. L.*)

Acute Bronchopulmonary Diphtheria.—Two cases are reported of acute primary tracheobronchial diphtheria without secondary laryngeal or pharyngeal involvement. Both patients were young adult males. In one, a fatal fulminating type of diphtheria developed; but in the other, the virulence was less severe. This patient recovered following the administration of large intramuscular and intravenous doses of antidiphtheria serum together with insulin and glucose to support the heart. Early diagnosis is of vital importance if adequate treatment is to be instituted, and it is believed that a large proportion of these cases can be recognized early by roentgenographical rather than by bacteriological examination. Stereoscopic projections of the chest in these cases reveal a fairly uniform, feathery opacity throughout most of the lungs but with greater density in a very wide central zone. In general, there is a tendency to radiate upward, laterally, and downward from the hilum, the latter two directions predominating.—*Roentgenography of Acute Bronchopulmonary Diphtheria in Adults, N. Toomey, Radiology, August, 1933, xxi, 130.*—(*T. O. R.*)

Pulmonary Abscess Following Tonsillectomy.—A case of pulmonary abscess following tonsillectomy is recorded. Inasmuch as the patient did not respond to conservative measures during a period of seven weeks, portions of the eighth and ninth ribs were resected and the abscess drained externally.—*Lung Abscess Following Tonsillectomy, F. T. Ranson & L. McGolrick, Brit. M. J., December 2, 1933, no. 3804, 1020.*—(*T. O. R.*)

Spontaneous Pneumothorax without Demonstrable Cause.—Spontaneous pneumothorax occurred in a 43-year-old man without a history of tuberculosis, who noticed a "stab" in the right chest while taking a walk. After a few hours he had to go to bed because pain in the right chest, cough and dyspnoea appeared. A needle was introduced into the right pleural cavity and air escaped, apparently under high pressure. Repeated aspirations were made within the next few days. A bloody exudate developed 14 days later which was also repeatedly tapped. An X-ray showed that the lung had contracted to a narrow band and that there was a basal exudate. An X-ray two months later showed normal lungs except for a bean-sized calcified node in the left first interspace. No apparent cause for the development of a spontaneous pneumothorax could be found.—*Spontanpneumothorax ohne erkennbare Ursache, J. Woll, Deutsche med. Wchnschr., September 22, 1933, lix, 1469.*—(*H. S. W.*)

Chronic Bilateral Spontaneous Pneumothorax.—A man of 49 years was well until seven years previously. His illness started with a cold which resembled "flu." Since this time he has complained of a cough and increasing dyspnoea. After two or three years the cough became productive. He received several courses of vaccine therapy with no resulting benefit. His general health gradually declined. He became extremely dyspnoeic and cyanotic on the slightest exertion. His sputum did not contain tubercle bacilli, was profuse, mucopurulent in character but not foetid. Physical examination suggested bilateral collapse of the upper lobes. This diagnosis was confirmed by roentgenograms and the introduction of a needle in each chest cavity. The pressures on the left side were positive 6, negative 2 and on the right side positive 4, negative 2. The patient died suddenly about ten hours after the intrapleural investigations. Permission for a necropsy could not be obtained. Because of the insidious onset of the condition, a slowly growing neoplasm suggested itself as a possible cause. —*Chronic Bilateral Spontaneous Pneumothorax (with Special Plate), J. Lewis, Brit. M. J., October 28, 1933, no. 3799, 779.*—(*T. O. R.*)

Calcification of Pleura and Splenic Capsule.—A gold-miner, aged 59 years, was admitted to Papworth with a history of four years' illness. An X-ray of the chest showed bilateral fibrotic disease and a sharply defined opacity in the lower left chest. Death occurred fourteen months later, following an attack of dyspnoea. Postmortem examination showed bilateral fibrocaseous disease of the lungs with excavation in the left upper lobe. The right parietal pleura was not adherent, but the left was calcified and adherent to the sixth and seventh ribs. The

heart was enlarged and the muscle fatty. The diaphragm was normal. The spleen was calcified on its diaphragmatic surface. The fact that the calcium content of the splenic plaque was less than that of the thoracic one suggests that it was more recent. Nothing was found to account for the apparent extrapleural deposition of calcium in the thoracic plaque. It was rather unusual that the left parietal and visceral pleurae were not adherent to each other. Since the diaphragm is not a continuous sheet, but has two weak spaces, the calcifying splenic lesion might be the result of direct spread.—*Case of Pathological Calcification, L. B. Scott & V. Cotton-Cornwall, Lancet, September 30, 1933, ccxxv, 755.*—(*E. H. K.*)

Pulmonary Emphysema.—Pulmonary emphysema is a constant sequel of arrested tuberculosis. The less severe cases present many symptoms which are frequently incorrectly diagnosed. Rest is the only effective treatment and, the later the condition is identified, the more rest is required. For roentgenological diagnosis it is necessary to have stereoscopic films with good penetration, a lateral plate, and a fluoroscopic examination. In addition to this, the roentgenologist must be familiar enough with thoracic anatomy to correctly interpret the films. In emphysema, the ribs are well separated. The diaphragm is depressed and its excursion restricted. However, the most characteristic manifestation is the dislocation of thoracic viscera. The pulmonary arteries may be displaced upward and the bronchial trunks in all the lobes widely separated. In the lateral plate, the anterior dark area extends in front of the heart shadow unless the pericardium is attached to the anterior chest-wall. Late manifestations of emphysema are blebs which are usually seen in the bases, a barrel-shaped chest, and posterior bowing of the spine.—*Pulmonary Emphysema Associated with Arrested Pulmonary Tuberculosis, K. Dunham, Radiology, September, 1933, xxi, 207.*—(*T. O. R.*)

Cervical Rib.—Left artificial pneumothorax was instituted in a woman of 31 years with unilateral pulmonary tuberculosis. A large pleural effusion developed which resorbed slowly leaving the left side of her chest collapsed and contracted. Four months later the patient complained of sensory and motor disturbances in her left hand and forearm. Roentgenogram revealed cervical ribs bilaterally. The band between the accessory and first rib was divided and the scalenus anticus severed. The patient's symptoms have improved considerably.—*Cervical Rib with Symptoms Due to Dropping of the Shoulder Caused by an Obliterative Pleurisy, E. R. Boland, Brit. M. J., December, 9, 1933, no. 3805, 1068.*—(*T. O. R.*)

Changes in Ribs Overlying Empyema Cavities.—The ribs overlying empyema cavities lose their normal contour and become relatively round and somewhat larger in cross-section. These changes result from the deposition of subperiosteal new bone on the inferior and internal surfaces of the ribs. Synostoses occasionally occur. The internal architecture of the ribs becomes altered. Four cases are reported. The transformation is apparently brought about by two processes: (1) an inflammatory response in that portion of the rib adjacent to the parietal pleura, and (2) a functional response to abnormal forces acting on the rib as a result of contraction of the pleural scar. The latter force would cause such changes in the inner architecture as were observed.—*Ribs Overlying Empyema Cavities: Pathologic Study, J. D. Bisgard, Arch. Surg., 1933, xxvii, 941.*—(*R. D.*)

Heart in Pulmonary Tuberculosis.—In a roentgenological consideration of the heart in pulmonary tuberculosis, 750 consecutive tuberculous cases were studied. In 42.3 per cent, the X-rays revealed an abnormal appearance of either the heart or the aorta. It should be noted, however, that almost one-third of the observed patients were over 45 years of age and many of them engaged in strenuous work. Microcardia or cardiac displacement was observed in the majority of cases. In 8.6 per cent organic heart changes were diagnosed but valvular lesions were found in only 3.6 per cent. With the exception of right ventricular enlargement, most patients with organic heart changes showed minimal or moderately advanced tuberculous involvement.—*The Heart in Pulmonary Tuberculosis: a Roentgenologic Consideration, E. A. Schmidt, Radiology, August, 1933, xxi, 167.*—(*T. O. R.*)

Dermoid Cyst of Mediastinum.—Dermoid cysts of the mediastinum may give rise to pulmonary haemorrhage constituting the so-called pulmonary type. The cyst may either so encroach on the pulmonary parenchyma that mediastinal symptoms are masked by the development of purely pulmonary symptomatology, or less frequently, by a rupture of its wall give rise to the same group of symptoms. The latter contingency may follow trauma, but more often is the result of inflammatory reactions about the

cyst. The right lung is generally involved, the left ordinarily remaining unaffected. Dermoid cysts giving rise to haemoptyses usually occur in the earlier decades, and menstruation in women lends itself to an earlier manifestation of pulmonary bleeding in this sex. Shock or infection may initiate the haemoptysis. Thoracic pain on the right may precede the haemorrhage by weeks or months. The haemoptyses resemble those of tuberculosis in amount, color and recurrence. They differ, however, in that they ordinarily stop more promptly, are quite irregular, and not infrequently have long periods of remission. The blood may at times be brown rather than bright red due to stasis in the bronchi. The differentiation from tuberculosis is made on the failure to find the tubercle bacillus after careful search by smear, culture and guinea-pig inoculation. The positive diagnosis of a dermoid cyst can only be made on the demonstration of hair, even two or three, or of sebaceous material, verified by a test with osmic acid, in the sputa. Cells are not present in the expectorated material unless secondary infection or metastasis has occurred. Although examination by X-ray may be helpful in establishing a diagnosis, inflammatory reactions about the cyst may mask the true condition. The presence of a rounded mass in the upper mediastinum is suspicious. Cures result only with encystment or calcification of the teratoma, rarely as the result of surgical interference due to the dangers of pleuropulmonary complications and persistent bronchial fistulae. Death results from pulmonary infections, cachexia and metastases. Exitus rarely follows an haemoptysis. A case is appended illustrating the pulmonary type of dermoid cyst with pulmonary haemorrhages.—*Kyste dermoide du mediastin. Forme pulmonaire à "hemoptysies." Terminaison par encéphalite septique métastatique, G. Caussade, J. Decourt and A. Duroisel, Arch. méd.-chir. de l'app. respir., 1934, viii, 258.*—(*V. T.*)

Primary Cancer of Lung.—A study of the literature reveals an approximately tenfold increase in the number of cases of cancer of the lung coming to autopsy within the last forty years, and a twofold increase in the last ten years. There is no convincing evidence however that this is a real increase in the incidence of the disease. This increase may be partly accounted for by (1) the increased life expectancy from 43 to 58 years during the last half-century; (2) the discovery of the tubercle bacillus which resulted in an increase of diagnosis of carcinoma of the lung in lesions where no tubercle bacilli could be found; (3) the X-ray and bronchoscope, which have greatly increased our diagnostic capacity; (4) the greater interest in primary carcinoma of the lung; and (5) the fact that many cases of primary carcinoma of the lung were previously considered as metastatic, due chiefly to Virchow's opinion, which is now no longer held by pathologists. On the basis of studies from the literature as well as on the 12 cases reported in detail by the authors in this paper, they consider, as the most significant symptoms, cough followed by constant pain, and expectoration of blood-streaked sputum accompanied or followed by dyspnoea. Physical signs result from a gradually increasing bronchial tumor leading to limitation of motion of the affected side, decreased breath-sounds distal to the bronchial obstruction, and dulness or flatness over the tumor. The growth of the tumor can be followed by X-ray. Bronchoscopic examination reveals a nonelastic "woody" or fixed bronchus. Biopsy of bronchial tissue establishes the diagnosis. The absence of tubercle bacilli in the bloody sputum and, later, the presence of tumor cells therein, are an aid in diagnosis. A classification of the disease from the pathological and therapeutic standpoints is given. The extent of distant metastasis depends on the invasion of the primary tumor into the pulmonary veins.—*Primary Carcinoma of the Lung with Special Reference to Incidence, Early Diagnosis and Treatment, A. J. Hruby and H. C. Sweany, Arch. Int. Med., 1933, lii, 497.*—(*M. B. L.*)

Primary Cancer of Bronchus.—A man of 50 years suddenly developed pain in both shoulders and back. This was followed by weakness and numbness in both lower extremities and complete retention of urine. Roentgenogram of the chest showed the heart displaced to the left but failed to give any clue as to the cause of this displacement. The patient's condition rapidly grew worse. He died one month after onset of his symptoms. At postmortem, secondary metastatic deposits were found in the vertebral canal at the levels of the fifth and sixth thoracic segments and at the first and second lumbar segments. The right lung contained a large tumor growing from the outer wall of a branch of the right bronchus. Microscopical examination revealed this tumor to be an adenocarcinoma of the small-cell type.—*Primary Bronchial Carcinoma with Secondary Vertebral Deposits, H. A. F. Rofe, Brit. M. J., December 16, 1933, no. 3806, 1116.*—(*T. O. R.*)

Cancer of Lung.—Primary carcinoma of the lung presents one of the most discourag-

ing of any of the problems of malignant disease. Its resistance to various forms of irradiation is known and it is admitted that until more is known concerning the cause and specific cure of malignant disease surgical extirpation offers the most promising way to obtain a permanent cure. Lobectomy does not carry a prohibitive mortality. The location of the malignant lesion determines the symptoms. There may be no symptoms until late in the disease. Cough, partial bronchial obstruction, haemoptysis or pain may be the first symptom. In the roentgen ray film the early lesion usually shows as a rounded dense shadow. The opacity and displacement of the organs may be out of proportion to the size of the neoplasm. Differentiation from tuberculosis must be made early. Bronchoscopy often yields positive evidence. The details of a case of bronchiogenic epidermoid carcinoma are here given. The lower lobe of the right lung was extirpated successfully. At the time of operation it was seen that the tumor had invaded the posterior mediastinum and pericardium. The patient died 10 months after operation.—*Primary Carcinoma of the Lung, R. H. Overholt, Amer. Journ. Surg., November, 1933, xxii, 181.*—*(J. F. B.)*

Pneumonectomy for Cancer of Lung.—Primary carcinoma of the lung, which almost always arises in a bronchus, constitutes between 5 and 10 per cent of all carcinomata. Up to the present time the prognosis has been almost uniformly bad because of the complete futility of any methods of treatment other than surgical excision. There is no record of permanently successful treatment by radiotherapy of a single case. Therefore, unless some entirely new general principle in the treatment of carcinoma is devised, the only method that at present can offer any hope is the wide surgical removal of the tumor and the surrounding tissue. The authors report a case which is among the first in which an entire lung has been successfully removed for carcinoma. The entire removal was done in a one-stage operation. The patient was a physician aged 48 and symptoms referable to the left lung had appeared about seven months before. Spontaneous pneumothorax had occurred with some temporary relief. Bronchoscopic examination finally made the diagnosis possible, the growth extending so far medially in the upper-lobe bronchus as to make it possible to save the lower-lobe bronchus. At operation nodules in the upper portion of the lower lobe made it imperative to remove the whole lung. Despite the fact that the hilum of the entire lung was suddenly shut off by a tight ligature, none of the signs or symptoms of pulmonary embolism appeared, although this sudden obstruction of the pulmonary artery by the ligature was analogous to embolism. Not the slightest change was noted in the character of the patient's respiration. Seven ribs, from the third to the ninth, were removed from the spine to the anterior axillary line, to permit the soft tissues of the chest-wall to collapse against the bronchial stump and obliterate as far as possible the pleural cavity. Six weeks later, the first and second ribs were removed in almost their entire length. Convalescence was quite satisfactory. Examination of the lung after its removal was encouraging, showing no evidence of any extension of the carcinoma beyond the original site. The whole tumor measured only about 1 cm. in the long diameter. The nodules left in the lung at operation were small abscesses that showed no evidence of carcinoma. The tumor itself was a squamous-cell carcinoma. A probably important feature was that it had not invaded the bronchial cartilage. In the light of the experience derived from this case the operation would seem to be one that is entirely feasible in properly selected cases. There is now little excuse for the common failure to diagnose a carcinoma of the bronchus in its early stages. The majority of such tumors can be diagnosed before demonstrable metastases have occurred.—*Successful Removal of an Entire Lung for Carcinoma of the Bronchus, E. A. Graham & J. J. Singer, J. Am. M. Ass., October 28, 1933, ci, 1371.*—*(G. L. L.)*

Chicken Serum in Hodgkin's Disease.—The method recently described by Utz and Keatinge was used in the treatment of 5 cases of lymphadenoma (Hodgkin's disease). The method was deliberately modified in one respect: while fresh Hodgkin's nodes were inoculated into chickens as described, the chicken serum was not used in its natural state after removal from the clot. Such sera were found frequently to be contaminated by a staphylococcus and the risk of infection is great. The serum was therefore diluted after collection with an equal volume of 0.5 per cent phenol in physiological salt solution and filtered through a Seitz filter. The patients were injected at intervals of seven days and the chickens were bled at intervals as required. None of the chickens suffered in health as a result of their treatment and showed no disease when they were later killed. The results were disappointing. Four out of the 5 cases in a varying degree gave sensitization reactions. Without exception, the cases either had been treated or

were simultaneously treated by deep roentgen rays. The progress of these patients does not appear to have differed from what might have been anticipated in the absence of serum therapy, apart from the serum sensitivity developed.—*Treatment of Lymphadenoma with Chicken Serum, R. J. F. Pulvertaft, Lancet, October 14, 1933, ccxxv, 857.*—(*J. S. W.*)

Chicken Serum in Hodgkin's Disease.—Four patients suffering from Hodgkin's disease were treated with chicken serum according to the method of Utz and Keatinge. The procedure detailed by these authors was suggested to them by the work of l'Esperance on the aetiology of lymphadenoma in which evidence was produced to show that it was a form of avian tuberculosis. The technique used was briefly as follows: A fresh portion of node removed from the patient was emulsified in a small quantity of physiological salt solution and injected subcutaneously into the leg of a chicken. Ten days later the chicken was bled and the serum prepared, subsequent bleedings being done at weekly or fortnightly intervals. None of the cases were benefited by the treatment, nor was the disease controlled in any way. The injection of foreign serum was not without danger as evidenced by thrombosis in one case and serious anaphylaxis in another.—*The Serum Treatment of Hodgkin's Disease, N. R. Barrett & L. T. Bond, Lancet, October 14, 1933, ccxxv, 855.*—(*J. S. W.*)

Syphilis of Lung.—*Case Report:* A man, about fifty-five years of age, slowly developed cough and expectoration and increasing dyspnoea. This ailment was preceded by mental disturbance of the maniac type. The roentgenogram of his chest showed scattered bilateral nodular and infiltrative lesions, which were most massive in the lower portions and absent in the apical fields. His Wassermann reaction was positive, but specific treatment did not bring about any improvement. The important necroptic findings were as follows: Chronic interstitial syphilitic pneumonia of both lungs with miliary gummata and extensive sclerosis. Chronic gummous and purulent bronchitis. Secondary emphysema. Marked hypertrophy and dilatation of the right ventricle. Syphilitic ulcers of the vocal cords and epiglottis. The histological examination of the lungs showed many miliary gummata in the alveolar and bronchial walls. In the lower lobes there were massive nodular lesions which were composed of the same type of miliary gummata and of sclerotic tissue. The extensive sclerotic lesions are interpreted as healed syphilitic lesions, aggravated by a concommitant pneumonoconiosis. The gummata showed much fatty change, to the extent that free fat was present between the scar-tissue fibres and in the sinuses of the draining lymph nodes. In similarity with other reported cases of pulmonary syphilis, extrapulmonary lesions were few and slight.—*Ein bemerkenswerter Fall von Lungensyphilis, K. Scriba & H. E. Büttner, Virch. Arch. f. path. Anat., December, 1933, ccxci, 571.*—(*M. P.*)

Hernia of Lung.—A miner, 30 years of age, sustained extensive injuries of the muscular and bony structures of his thorax. Eighteen months after the accident a herniation of the lung became evident. A persistent cough presumably resulted in weakening the scar-tissue in the chest-wall and pectoralis major. The hernia was adequately and comfortably controlled by a corset.—*Hernia of the Lung, A. B. Pain, Brit. M. J., January 13, 1934, no. 3810, 59.*—(*T. O. R.*)

Polycythaemia in Pulmonary Disorders.—The essential cause of polycythaemia secondary to pulmonary disorders seems to be chronic anoxaemia, which stimulates the bone-marrow to excessive erythropoiesis. The case reported is that of a Swede, aged 45 years, who had worked as a lumberjack for twenty-five years, the last six of which were spent at 10,000-feet altitude. For ten years he had had frequent attacks of asthma, becoming worse following pneumonia five years previously. Seven months before, while working at 10,000-feet altitude, cyanosis, weakness and dyspnoea were noticed for the first time. Exhaustion, epigastric distress, and headache aggravated by exertion were noticed four months later. Improvement followed rest at a lower altitude, but symptoms returned on exertion. Asthma increased and cough and expectoration (sometimes bloody) began. Cyanosis of the face and extremities and clubbing of the fingers were present. There were rhonchi and signs of emphysema over both lungs. The heart was enlarged and the pulmonic second sound was accentuated. The blood-pressure was 100 systolic and 80 diastolic. The pulse was 90, and the respiratory rate and temperature were normal. The liver was enlarged and tender. The spleen was not palpable. The urine contained albumen, hyaline casts and erythrocytes. The blood Wassermann was negative. The erythrocyte count was 6,100,000, with 80 per cent haemoglobin. The leucocyte count was 5,600, with 79 per cent polymorphonuclears. Against advice, work was resumed at 10,000-feet altitude. Six months later there were extreme cyanosis and oedema of the

feet. The blood examination showed 7,210,000 erythrocytes, 120 per cent haemoglobin, and 4,600 leucocytes. A chest roentgenogram revealed a slightly enlarged heart, enlarged pulmonary-artery arch, and advanced pulmonary fibrosis. An electrocardiogram showed right ventricular preponderance and changes indicating coronary disease. The basal metabolic rate was 60 plus. Blood sugar, urea, and non-protein-nitrogen were normal. The sputum was repeatedly negative for tubercle bacilli. Haemorrhage after tonsillectomy lowered the erythrocyte and haemoglobin values, but they rose again within a few days. Until the last few months of life the cycle of work in the mountains, relapse, hospital treatment at lower altitude, improvement, return to work, and relapse was followed. The erythrocyte count reached 8,370,000 and cyanosis and dyspnoea increased steadily. Somnolence was prominent. During the last four years of life, haemoptysis always followed work in the mountains. Rest at a lower altitude always caused a decrease in the symptoms, the erythrocyte count, and the size of the heart, and disappearance of abnormal urinary findings. The case was reported from the Mayo Clinic as *polycythaemia vera* with a pulmonary complication either unrelated or secondary to pulmonary stasis. After two courses of phenylhydrazine he was discharged as improved, with a lowered erythrocyte count. The conjunctivae were bluish-red, due to a myriad of dilated blood vessels, and the vessels of the fundi were very tortuous and dilated. Many fine, round, scintillating floaters were seen in the vitreous of the right eye. Despite rest at sea-level, digitalis, venesections, phenylhydrazine, and radiotherapy of the long bones, death occurred with extreme oedema of the body tissues and the lungs, with signs of cardiac failure and terminal pneumonia. In the diagnosis it was necessary to consider (1) polycythaemia vera with a pulmonary complication, (2) Ayerza's disease, and (3) polycythaemia of high altitude. Against the diagnosis of polycythaemia vera are cited (1) the chronic pulmonary disorder and dysacclimatization, themselves sufficient cause for anoxaemia; (2) the presence of cyanosis instead of erythrosis; (3) decrease in the erythrocyte count and the size of the heart after rest at a lower altitude, and increase in the blood count with dilatation of the heart after work at 10,000-feet altitude; (4) the normal-sized spleen; (5) leucopoenia instead of leucocytosis; (6) the absence of qualitative changes in the blood cells; (7) dyspnoea; (8) hypertrophy of the right ventricle; (9) the perfect clinical picture of chronic mountain sickness; and (10) somnolence, which is frequent in both Ayerza's disease and the erythraemia of high altitude. The reason why a case of pure polyglobulism should have cyanosis or dyspnoea is not clear, but there is every reason why a case of chronic cyanosis and dyspnoea should have a chronic erythraemia. Probably all erythraemias associated with cyanosis and dyspnoea are compensatory in nature. The high blood-volume is in favor of polycythaemia vera, though moderate increases occur normally in response to high altitude. Plethora and cyanosis have been found associated with tuberculous pericarditis and congenital pulmonary stenosis. The essential features of the case reported correspond exactly with those of Ayerza's disease. Erythraemia of altitude differs from Ayerza's disease chiefly in that in the former there is absence of sclerosis of the pulmonary arteries and complete relief occurs on residence at sea-level. Failure of the mechanism of adaptation to high altitude may be due to a diminution in the pulmonary minute ventilation. In chronic mountain sickness there is a chronic compensated gaseous alkalosis in contrast to a chronic compensated gaseous acidosis in Ayerza's disease. The oxygen tension of a "black cardiac" may be dangerously close to the critical level at which cardiac glycogen will be depleted and cardiac failure ensue. The vital capacity, tidal air, and oxygen saturation of the arterial blood are also reduced. Ordinary emphysematous cases are distinguished by a greater tolerance for high carbon-dioxide percentages. In the indigenous person acclimated to altitude the basal metabolic rate is normal. In acute mountain sickness it is below normal. In severe erythraemia of altitude and chronic mountain sickness it is high. In severe erythraemia of altitude the high basal metabolic rate creates a demand for oxygen which is satisfied only by return to sea-level, and anoxaemia occurs readily. Anoxaemia causes the normal mammalian heart to dilate. The sequence of events in the reported case was reconstructed as follows: Long-standing bronchial asthma and emphysema produced a low-grade anoxaemia which was aggravated by severe exertion at high altitudes. An excessive response of the bone-marrow to the stimulus of chronic anoxaemia induced a high grade polycythaemia. The increased volume of abnormally viscous blood and the reduced pulmonary capillary bed caused cardiac strain. The metabolic rate rising with increased marrow activity magnified the bad effect upon the heart of increasing anoxaemia. Periodic cardiac decompensation on visits to high altitude produced rhythmic aggravation of

anoxaemia which stimulated erythrogenesis. Necropsy revealed hypertrophy of the heart, especially the right ventricle, slight subacute myocarditis, slight sclerosis of the pulmonary artery, with generalized dilatation of the pulmonary arterial system, chronic bronchitis, emphysema, acute but not extensive bronchopneumonia, extreme subacute glomerulonephritis, moderate hyperplasia of the bone-marrow, chronic adhesive pleurisy, and dilatation of the veins of the splanchnic region, especially in the liver. Literature concerning Ayerza's disease is discussed. It is quite clear that syphilis is frequently present in the "black cardiac," but it is not clear that the arterial lesion commonly found is syphilitic. The physiological effects of the various pathological conditions in the reported case are analyzed. Roentgenograms of the chest and photomicrographs of tissues are reproduced.—*Polycythemia in Association with Pulmonary Disorders, J. J. Waring and W. B. Yegge, Ann. Int. Med., August, 1933, vii, 190.*—(*R. M. F.*)

Tuberculosis and Poliomyelitis.—During 12 years at the Kharkov Medical Institute where over 4,000 cases of tuberculosis and 1,000 cases of infantile paralysis have been seen, only one case has been observed in which there was undoubted coincidence of the two diseases. A boy aged 14 was well until three years old. Following an injury, and a repetition of it four months later, he was unable to use the left leg. After three months in a plaster cast the patient was able to walk. At this time he developed an acute illness with high temperature. Six weeks later it was discovered that both legs were paralyzed. The use of the right leg returned gradually, the left remained paralyzed. At present the left leg shows marked atrophy of the muscles, eversion of the hip, genu valgum, with foot in equinus. There is 13 cm. shortening. All active movements are absent. Passive movement of the joints is free. X-ray examination shows upward dislocation of the hip, the acetabulum widened and deformed, head of the femur absent and very slight attempt at repair. The Pirquet reaction is positive. There is some residual paralysis of the right leg muscles.—*Tuberculosis and Poliomyelitis, E. Rumshina, Jour. Bone & Joint Surg., July, 1933, xv, 772.*—(*L. F. B.*)

Rheumatic Lung.—That the rheumatic virus may attack both lung and pleura has been known almost two hundred years. The incidence of pleural and pulmonary manifestations has been reported as 10.5 per cent in rheumatic endocarditis, 58 per cent in rheumatic pericarditis, 71 per cent in rheumatic endopericarditis, and in from 0.64 per cent to 9.94 per cent in rheumatic fever. Cheadle described pleurisy in rheumatic fever as occurring either at the time of the arthiritis or toward the end of rheumatic heart disease. The association of pleurisy with rheumatic manifestations has been reported in from 2 to 40 per cent of cases. Next to carditis, it is the most common complication of rheumatic fever. The nature of the pleurisy was first described by Paul in 1928. First, a thin film of fibrin appears on the pleural surface. Later this becomes thicker and adhesions are organized. The thick, hyalinized pleura of tuberculosis does not result. Histologically, the lesion is characterized by desquamation of the endothelial cells, accompanied by severe, chronic, nonsuppurative inflammation throughout the subpleural layers. Rhea found structures resembling Aschoff bodies in one case. Usually there is an effusion when there are extensive mediastinitis and pericarditis. Paul concluded that the pleurisy is part of the generalized process and not the result of direct extension from the pericardium. Extensive involvement of the pulmonary arteries and arterioles and the presence of Aschoff bodies in the adventitia of the pulmonary arteries and of the aorta have been described. The arterial endothelium seems to be involved primarily. The question of the existence of a specific rheumatic pneumonia is undecided. The pulmonary lesions have been thought to consist of oedema, atelectasis, and passive congestion rather than a true exudative process. Consolidation does occur in the right upper lobe, however, and this lobe is relatively free from the influence of cardiac failure. Naish found that the consolidated lung of rheumatic fever is not friable, has the consistency of india-rubber, is of a homogeneous purplish-red color, and has none of the marble appearance of other pneumonias. He was impressed by the extent of consolidation, involving nearly all of the five lobes in four of his six cases. Of Coburn's 3,000 rheumatic patients, 30 suddenly developed consolidation without evidence of congestive heart failure. The rapid disappearance and migratory nature of the areas of consolidation were characteristic. Gouley and Eiman described the acute pulmonary lesion as an acute interstitial inflammation, having as its basis the vascular damage and perivascular infiltration common to all rheumatic lesions. The perivascular interstitial infiltration of large cells, often multinucleated, is the significant histology. Later large cells with solid, irregular nuclei replace the large Aschoff cells, indicating subacuteness and early sclerosis. The pneumonia is not

preceded by upper respiratory-tract infection, there is no chill, and the respiratory rate is only slightly increased. Virulent pneumococci are absent from the sputum. The striking clinical feature is the transient character of the physical signs. Few râles are present during consolidation. Later coarse pleuritic râles are heard.—*The Rheumatic Lung, C. P. Howard, Ann. Int. Med. August, 1933, vii, 165.*—(*R. M. F.*)

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